

ANEURISM OF AN ANOMALOUS ARTERY CAUS-
ING ANTERO-POSTERIOR DIVISION OF
THE CHIASM OF THE OPTIC NERVES
AND PRODUCING BITEMPORAL
HEMIANOPSIA.¹

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NOTE-BOOK, Case No. 836, D. G. H., æt. 43, manufacturer of bromine, from the Springs in Kentucky, consulted me May 29th, 1886. He was a large, florid, wholesome looking man without previous grave disease. He had had for thirty years hæmorrhoids, which at times bled freely. All of his functions, digestive, secretive and reproductive, seem to have been perfect. Of late, he had felt fatigued from any unusual exertion; also, the legs and arms became easily numb in sleep, or from pressure or malposition when awake. He has had for a year varying but gradually increasing pain in the parietal and vertex regions, and at times this pain darts through to both temples or to either. Excessive exertion causes it to increase if present, or may bring it on.

To shake the head and jar it causes no pain.

Hearing good. At no date had he any noises in the head.

Vertigo none. Station normal, with eyes open or shut. Knee-jerk normal.

Olfaction natural; but not examined later in the case.

He says that three years ago, during very hot weather, he became abruptly weak in the legs, so that he fell on his knees and hands, but did not lose consciousness. For a few hours his right foot dragged, but he had been able to rise and walk, and had no other annoyance than great sense of fatigue.

¹ Read at the meeting of the American Neurological Association, Washington, D. C., September, 1888, by Dr. F. X. Dercum.

The following is the report of Professor William Thomson of the state of the eyes in Mr. H.'s case :

"On May 29th, 1885, when first examined, the patient stated that eighteen months ago he began to have trouble with his sight in the form of diminished vision in the left eye, particularly towards the left side, which gradually increased, and that six months later the right eye began to fail in vision towards the right side. The acuteness of vision is R. E. $\frac{6}{60}$, L. E. $\frac{6}{60}$, equals $\frac{1}{10}$, and at 20 centimetres, he reads $D = 2$, showing same acuteness. Has full power of accommodation, normal sensibility of each iris to light and perfect fixation of each eyeball and consequent integrity of each third nerve. Sensibility, taste, smell and hearing are normal. He has entire loss of sight to the right of the vertical line with the right eye, and to the left of the same vertical line with the left eye, showing complete anæsthesia of the nasal half of each retina. This was carefully determined by the use of the two lights of my ametrometer and was accurately vertical for the left eye, but inclined slightly in the right eye, being 10° to the right in the upper and 5° to the left in the lower part of the field. The color sense was yet good in the remaining half of each field, and he recognized promptly red and green when the lights were tinted with glasses of these colors.

By ophthalmoscopic examination the fundus presented no changes except at each papilla, where the vessels of the retina appeared perhaps somewhat attenuated, whilst the neuroglia, especially of the left eye, was pale, the porus opticus enlarged, and the appearances those of partial atrophy. There was no swelling of either papilla, nor any change in the retina that would indicate a previously "choked disc."

The diagnosis was pressure at or in front of the chiasm, sufficient to cut off the connection between the inner halves of each retina, with neuritis or partial atrophy, especially of the left nerve to account for the low acuity of vision, caused by some growth which did not excite enough meningitis to cause a swollen papilla.

On November 16th, 1885, the only change observed was an increase of acuteness in the right eye to $\frac{1}{3}$, being for the

R. E. $\frac{6}{18}$ and for the L. E. $\frac{6}{60}$, with more marked appearances of atrophy in the left papilla. The fields of vision were carefully determined by the perimeter, *vide* Figs. 1 and 2. He was found to have slight astigmatism in each eye, but this was disregarded, and I gave him $+1.5 \frac{Rt.}{L.}$ for his near vision, with which he could read with the right, D = 050 at 25 centimeters.

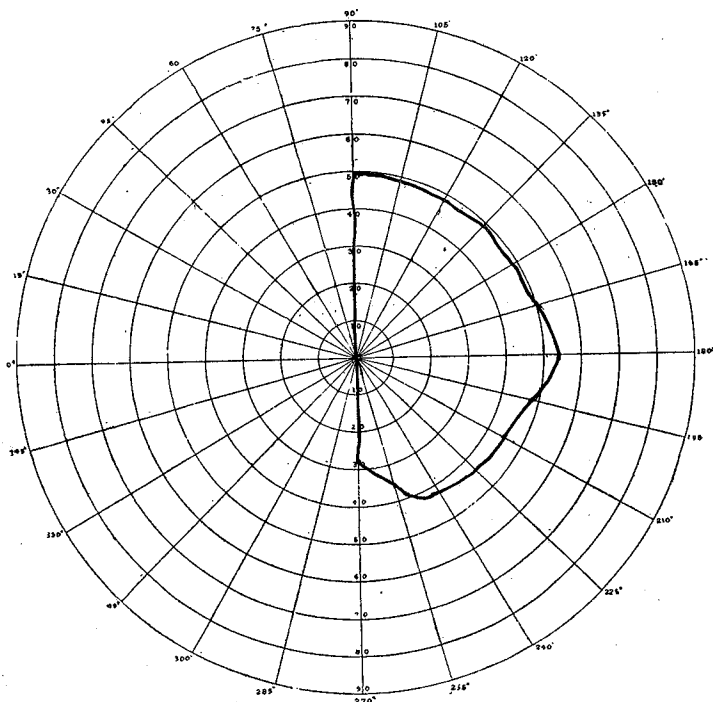


Fig. 1.—Illustrating Field of Left Eye.

June 16th, 1886, R. E. V. = $\frac{6}{18}$, L. E. $\frac{6}{60}$ by excentric fixation, the hemiopia being the same, but difficult now to determine on the left, except with lights.

Feb. 7th, 1887, R. E. V. = $\frac{6}{18}$, L. E. $\frac{6}{30}$ only; hemiopia as before, pupils responsive, dilated with cocaine, both papillæ more white, retinal vessels normal and full in size. Although the left was now reduced to $\frac{1}{30}$ only, the hemiopia was readily detected with the ametrometer."

In February, 1887, Professor Harrison Allen examined the nose, and found much discharge of thin mucus. He

were aware of the deep interest taken in this case by Professor Thomson and the author. They made, therefore, earnest efforts to secure an autopsy and at last succeeded. Without them the instructive lesson of this case had been lost. I append here the notes of the cadaveric section as made by Dr. McLean.

"Autopsy, May 24th, at 8 A. M., seventy-five hours after death: Rigor mortis slight. External appearance shows marked purple discoloration of both ears, with large purple blistered surface on back of neck. Otherwise appearance of one dying in apparent perfect health. Weight 200 pounds. Eyes: pupils widely dilated, with marked congestion of all conjunctival vessels, ocular and palpebral. All the vessels of the scalp and external surface of calvarium, markedly congested. Dura mater not adherent except on inner surface along each side of longitudinal sinus, at which points slight exudation of lymph was found. All vessels of the pia mater of a deep purple color and in a state of extreme congestion. Nearly fourteen ounces of serum escaped during removal of the brain.

Elevating the anterior lobes of the cerebrum, the olfactory nerves and bulbs came into view and also the optic nerves entering the optic foramenia. The optic nerves were widely separated (fully one inch) by a large purple tumor filled with fluid, lying directly between them. About half an inch of each nerve could be traced backward from the optic foramen on each side of the tumor, which came up out of the sella Turcica and evidently by pressure had caused absorption of the olivary process and the optic groove as far as its extreme anterior border. A separation seemed to have taken place in the centre of the optic commissure, pushing the optic nerves and tracts to the outside of the tumor. The commissure could not be found. In attempting to dissect the tumor from the deep cavity in the sella Turcica, the upper rounded extremity slipped from its cavity in the brain, the centre of which cavity could be marked by a point directly in the centre of the circle of Willis. The right and left internal carotids were found

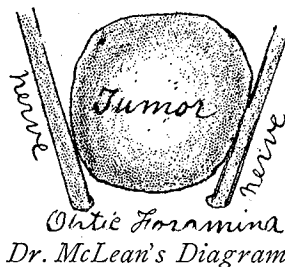
intimately connected with and apparently forming the tumor. They were divided by the knife at its upper border just before giving off the posterior communicating branches. The entire brain was now removed, leaving the tumor deeply imbedded in its cavity in the sella Turcica. The tumor was in no way attached to the brain except by the internal carotids, but was firmly attached to the bone around the border of the deep cavity which it had formed in the sella Turcica. The tumor being removed, the bony cavity was found quite rough and honey-combed. No further examination was made of tumor or brain, but both were immediately forwarded to Drs. Thomson and Mitchell.

Signed,

J. T. McLEAN."

So anxious was I as to the accuracy of the examination that I asked in a letter, if the chiasm could possibly have been torn during the autopsy. To this query Dr. McLean replied as follows:

"The optic nerves were not torn. As the anterior lobes of the cerebrum were gently and gradually lifted, the olfactory nerves and bulbs being in place and perfect, the optic nerves came into view. By the aid of a rough dia-



gram, I perhaps can make it plain to you what I saw, viz., what seemed to be the neck of a large purple tumor coming up from the sella Turcica and pressing far forward directly between the anterior clinoid processes and directly between the optic foramina. The optic nerves were plain and distinct and perfectly straight on each side of the tumor, entering the optic foramina, and I noticed the peculiar direction, viz., widely separated and slightly converging, as here represented.

"Gently lifting anterior lobes in order to get more room to work, the large upper bulbous portion of the tumor slipped from the cavity in the cerebrum, as heretofore described. If it were possible for the commissure to have been pressed far behind the tumor, it might have been torn when the upper rounded part of the latter slipped out of the cavity it had formed in the cerebrum, but no trace of the commissure could be found, but only the two long straight optic nerves and tracts, as you no doubt observed in the specimen."

The specimen as it arrived was examined by the author with Professor Wm. Thomson and Dr. F. X. Dercum, and all were fully satisfied as to Dr. McLean's accuracy. The following study of the parts sent was made by Dr. Dercum, to whom is due also the collection of anomalies.

The case speaks for itself, as it stands alone in the records of pathological accidents. It seems needful to suppose that an anomalous artery connected the carotids by passing under the chiasm. This branch became aneurismal and enlarging, lifted the chiasm until this parted in the middle line, leaving a nerve on each side, thus dividing the right and left fibres which, crossing in the chiasm, supply the nasal sides—the temporal visual fields of each eye. At what date the final division took place it is impossible to say, nor does the ocular study made by Professor Thomson settle this point, since as early as the first examination of the eyes there was complete anæsthesia of both nasal halves of the two retinas. The left eye had evidently suffered most, and as time went on continued so to do. The absence of choking or optic neuro-retinitis is a notable fact, and that the presence of a pulsating mass as large as a lemon, caused so little disturbance of mind or of motor or sensory functions is interesting.

If any doubt remained as to the uses and functions of the internal bandelettes, this case assuredly settles the question in a most decisive fashion. It is a slowly done vivisection, effected with the least possible disturbance by a pathological process.

I leave any further discussion to my friend, Dr. Dercum.

When the brain was examined by Drs. Mitchell, Thomson and Dercum, it was floated in alcohol with its base upward. It was noted at once that the various structures normally occupying the space included by the crura and the basal portions of the frontal and temporal lobes were entirely wanting. No trace of an infundibulum or a chiasm was discoverable. In their stead, a huge cavity extending deeply into the brain was found. The walls of this cavity were irregular, softened, and disorganized. Lying to either side of it were revealed two white and rather ragged bands. These evidently corresponded to the optic nerves and tracts described by Dr. McLean in his record of the autopsy. Their anterior portions certainly looked like the optic nerves, but as they were followed backwards they became attenuated and ragged. In fact nothing but the anatomical relations of the posterior portions could justify one in calling them the optic tracts. The nerve on the right side, though imperfectly, was the best preserved; that on the left side was exceedingly difficult to follow, for, in the region of the destroyed chiasm, it was reduced to a few mere shreds. In addition, both nerves were so soft and brittle that the most careful handling caused them to break and tear.

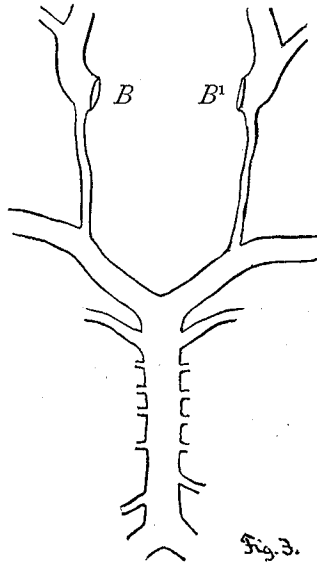
The circle of Willis was next carefully dissected. It is represented diagrammatically in the accompanying figure.

The openings B and B¹ communicated directly with the aneurism about to be described, *i. e.* judging from the relation of the parts and the accurate account of Dr. McLean. The tumor, though evidently shrunken by the alcohol, was decidedly larger than an egg and pyriform in shape. It had been ligated at the smaller end with a thread, and when originally examined was distended with fluid, and was of a dark, purple color. The larger portion, or base, fitted readily into the cavity of the brain already described; in other words, the larger end had been directed upwards, while the smaller end had occupied the sella Turcica. Upon handling a quantity of blood oozed out notwithstanding the ligature.

Careful dissection revealed two large openings at the smaller end. These communicated freely with the interior,

and were so directed that they faced slightly downwards and to the right and left respectively.

It requires no great effort of the imagination to conceive of these openings as originally confluent with the openings B and B¹ in Fig. 3. Indeed this explanation becomes more



probable when we reflect that the condition of the parts admits of no other. No other opening was detected in the cavernous carotids, and no other anomalous vessels were present in the circle of Willis from which the aneurism could arise.

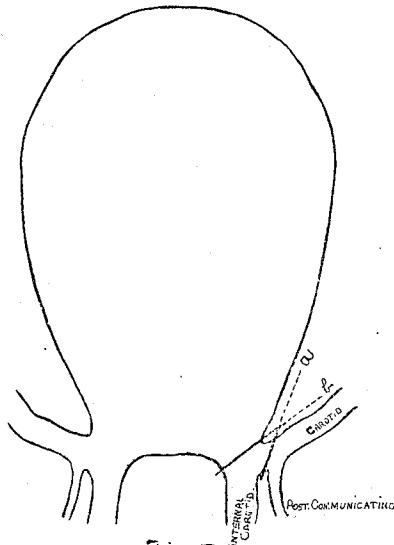
A difficulty, notwithstanding, suggests itself. What has become of the remaining or pre-circular portion of the encephalic carotids? Naturally we should expect it to be adherent either to the circular portion of the carotid or, if severed from the latter, adherent to the aneurism. Evidently and very naturally, Dr. McLean in removing the tumor practiced two incisions. First, he separated the tumor from the carotids at a point "just before giving off the posterior communicating arteries." The doctor further tells us that, on removing the brain after this division, the tumor remained "deeply imbedded in its cavity in the sella Tur-

cica," and also that "the tumor was in no way attached to the brain except by the internal carotids, but was firmly



Fig. 4.

attached to the bone around the border of the deep cavity which it had formed in the sella Turcica." He therefore practiced a second incision in the final removal of the



tumor, thus severing it from the pre-circular carotid. See Fig. 5, which shows the probable relation of the parts and the direction of the incisions. Line *a* represents the direction of the first cut, following which the brain together with the circle of Willis was removed, leaving the tumor still in the cranial cavity. Line *b* represents the cut which finally separated the tumor from the remaining portion of the carotid.

An attempt was made to study the optic nerves and tracts and the two cunei microscopically; but owing to the softened condition of the structures, the attempt was not successful. The parts had been macerated during the journey in an insufficient amount of alcohol, and post-mortem changes had evidently taken place.

The remnants of the nerves and tracts revealed nothing definite, except perhaps a relative excess of connective tissue elements. The cunei also showed no decided evidences of pathological changes, except perhaps the left. The latter showed here and there marked dilatation of the pericellular lymph spaces; yet whether these appearances indicated cystic degeneration, or were merely the outcome of a stronger alcohol acting upon an already softened cortex, is problematical.

In order to throw additional light upon this remarkable case, a collection of anomalies of the circle of Willis was made, excluding of course abnormalities of the basilar and pre-circular portions of the carotids.² This study has not proved without benefit. In explaining this case, Dr. Mitchell assumes the existence of an anomalous vessel connecting the two carotids. That such a vessel did exist, there can I think be no doubt. In fact, that there is a general tendency to the anastomosis of the opposite sides of the circle of Willis is shown by the great constancy of the normal anterior communicating artery as well as by its anomalies. Regarding the latter, there may be many varieties. We may have, for instance, instead of one vessel, two. Incoronato and Barbieri record such cases, and Spitzka has given me the notes of a third. These instances of double anterior com-

² See Appendix.

municating artery are very significant. They present, as it were, an exaggerated expression of the normal tendency to anastomosis. Again, it surely makes but little difference in the functional value of the anterior communicating artery just at what point it crosses the circle, whether it connects the anterior cerebrals high up in their course or low down. After all, it is only a question of the relative position of the transverse vessel as to whether it can influence by aneurismal disease the optic chiasm. If it be sufficiently low down, interference with the chiasm becomes inevitable.

In order to show that Dr. Mitchell commits no breach of anatomical probability in supposing an anomaly to have existed, it may be stated that almost every conceivable variation has been known to occur. Thus, to begin with, the anterior communicating artery may be entirely wanting. Barbieri and Spitzka have each reported such cases. Again, the vessel may be present, but it may be impervious (Barbieri), or so small as to be filiform (Barbieri), or it may be unusually short (S. G. Weber, Barbieri), or it may be abnormally large in diameter (Barbieri), or it may be both abnormally short and abnormally thick. Again, one carotid may furnish both anterior cerebrals (Beaumont, Randall), or the anterior cerebrals may be undivided, that is, they may be represented by a single longitudinal vessel formed by the two carotids, just as the basilar is formed by the two vertebrals (Ebstein). Finally, *there may be a direct anastomosis of the carotids without the intervention of any communicating vessel*. Incoronato described a beautiful instance of such a case. The anterior cerebrals form a large quadrate lacuna or sinus, before they separate.

We have, therefore, in the anomalies of the circle of Willis, especially in its anterior portion, abundant evidence of its variability and abundant justification for the explanation by Dr. Mitchell that we have here an aneurism of an anomalous vessel.

Another point requires passing notice. Regarding division of the optic chiasm by the aneurism, doubt might for a moment arise in our minds as we recall the fact that the optic commissure has been, on some rare occasions, found

wanting. In such a case the optic nerve runs to the eye of its own side without any exchange of fibres with its fellow. That such was not the case in the present instance is shown by the condition of the optic nerves and tracts already detailed. They had undergone marked destructive change, especially in the region corresponding to the chiasm. It is exceedingly improbable that much, if any, destructive change would have occurred in the absence of a chiasm. The nerves would in the beginning have been widely separated, and an aneurism growing between them would in all likelihood have merely displaced them. The clinical history, too, of sharply defined hemianopsia, utterly discounts this view.

Regarding the manner of the division of the chiasm, it is extremely probable that it occurred from below upwards and from before backwards. A study of the anatomical relations of the parts and of the probable position of the anomalous vessel, make this the only tenable view. Besides, it can be readily understood how gradual and long-continued pressure applied to the chiasm in this position would lead to destruction of the internal bands, and, as a consequence, to the eventual separation of the nerves. That the pressure was not exactly in the middle of the commissure is rendered probable both by the clinical history and by the unequal destruction of the two nerves and tracts.

Excessive care has been taken in the examination of the case, and the utmost caution in our inferences. If, as we believe, our pathological explanation be correct, this case stands alone in pathogeny. Moreover, it is an absolute demonstration of the function of the internal bandelettes in accordance with the views of modern pathologists.

APPENDIX.

A Collection of Anomalies of the Circle of Willis.

Barkow, Blutgefäesse der Menschen, 1866. The anterior communicating artery may be double or may make a plexus, or may give off an accessory anterior cerebral.

Barkow, Schlagad. der Saugethiere, 1866, Taf. 43. The anterior cerebral arteries unite like the vertebrals to form one anterior trunk like the basilar.

Spitzka, communicated by letter [not separately published].

a. Anterior communicating artery absent.

b. Left posterior communicating artery absent.

c. Anterior communicating artery double.

Decker, Sitzgsberichts der Würzb. Phys. Med. Gesellschaft vii., Stzg. No. 3, S. 33-43. [Virchow-Hirsch.] describes an anomalous vessel springing from the cavernous carotid and joining the basilar. Both vertebrals are exceedingly small, but both posterior communicating, very large and form the main supply of the basilar. They are also directly continuous with the posterior cerebral.

Barbieri, Agostino, Monografia della arteria vertebrale, Milano, 1867-68. The following brief abstracts were made from twenty cases collected by Barbieri:

CASE 1.—Impervious anterior communicating artery. Basilar artery nine m. m. in size. Branches of the carotid comprise the posterior communicating, which are rather pronounced.

CASE 2.—Short and large anterior communicating.

CASE 3.—Absence of anterior communicating. Anterior cerebral and posterior communicating on left side a little larger than ordinary. The posterior cerebral of left side smaller than the right and having the appearance of a double origin, in part from the internal carotid and in part from the basilar.

CASE 4.—Double anterior communicating. Left anterior cerebral natural, and right anterior cerebral a third larger than normal. Left posterior cerebral originates equally from internal carotid and basilar. Right posterior cerebral comes in greatest part from internal carotid. Both are very large.

CASE 5.—Anterior communicating short and large. Left anterior cerebral much larger than normal. Right anterior cerebral scarcely a little smaller than normal.

The posterior communicating arteries are merely branches extending backward from the carotid and after

being joined by small branches from the basilar they continue on in the ordinary course of the posterior cerebrals.

CASE 6.—Anterior communicating filiform. Left posterior communicating is of mixed origin arising both from carotid and basilar.

Right posterior communicating arises from carotid and communicates with basilar by means of an excessively slender twig.

CASE 7.—Anterior half of Circle of Willis well developed. Anterior communicating of ordinary length, but a little greater in calibre.

Right posterior cerebral originates from right carotid and communicates by a thread-like posterior communicating with the basilar.

Left posterior communicating, filiform and impervious.

Left posterior cerebral given off by the basilar.

CASE 8.—Anterior communicating normal. Right posterior communicating slender. Left posterior communicating normal.

CASE 9.—Anterior communicating normal. Anterior half of Circle of Willis better developed than the posterior. Right posterior communicating slender, and left posterior communicating a little larger than normal.

Left posterior cerebral originates equally from the carotid and the basilar.

CASE 10.—Circle of Willis as a whole small. Superiority of carotid over vertebral branches. Left posterior communicating quite contracted. Posterior cerebrals are equal in first part of course, but after being joined by the posterior communicating, the right receives a greater blood current than the left.

CASE 11.—Circle of Willis assymetrical through shortening of its left half. Anterior communicating large and short. An abnormal branch runs from anterior communicating forward and constituting a third anterior cerebral and equal in calibre to the other two [Wilder's "Termatic"?].

Right posterior communicating long, thin and impervious.

Left posterior communicating quite short and a little larger than normal.

CASE 12.—Both posterior communicating filiform.

CASE 13.—Both posterior communicating filiform. Carotid branches more pronounced than the vertebral branches.

CASE 14.—Anterior communicating very short and large. Assymetry of Circle of Willis. Left side complete and much developed. Right side incomplete and poorly developed.

Right anterior cerebral constricted previous to giving off of anterior communicating; thence normal in size.

Left anterior cerebral one-third larger than normal previous to giving off of anterior communicating and continues to be large afterward.

Right posterior communicating wanting. A minute twig springs from the right posterior cerebral at the part at which the posterior communicating is given off and extends for about two-thirds the usual course of the latter, when it breaks up into a number of minute branches. These are lost in the pia mater and brain tissue with the exception of a few that anastomose with small capillary branches given off by the right carotid.

Left posterior communicating, normal.

CASE 15.—Circle of Willis poorly developed, especially in posterior half.

Anterior communicating quite large and short. Left posterior communicating wanting, the right filiform.

CASE 16.—Anterior communicating slightly shorter and larger than usual.

Left posterior communicating wanting, replaced by minute branching twigs. Right posterior slightly narrowed.

CASE 17.—Anterior communicating of normal calibre, but rather short.

Right posterior communicating wanting, replaced by minute twigs.

Left posterior communicating larger than ordinary.

CASE 18.—Anterior communicating quite large and rather short.

Absence of both posterior communicating, small twigs in their places.

CASE 19.—Absence of posterior communicating, replaced by small twigs.

CASE 20.—Absence of left posterior communicating and filamentous condition of the right. Extremely filamentous anterior communicating.

Anterior cerebrals and posterior cerebrals very small.

Beaumontoir, Le Progrès Médical, 1886, p. 191, describes three anomalies:

1. The basilar furnishes a right posterior cerebral, but does not furnish a left posterior cerebral. This is supplied by the left internal carotid.

2. Left internal carotid furnishes a posterior cerebral, and this anastomoses with the basilar. It also gives off the middle cerebral, but no anterior cerebral. This comes from the right internal carotid. A minute vessel forms an anastomosis between the left carotid and the left anterior cerebral.

3. The right internal carotid gives off a middle cerebral, which, after a course of 18 m.m., divides into two branches which constitute the right and left anterior cerebrals.

Flesch, Verhandlung der Phys. Med. Gesellschaft zu Würzburg, x.—Virchow-Hirsch, Jahresbericht, 1886, ii. 12, describes a case in which the circle of Willis remains incomplete because of the absence of the posterior communicating artery.

Randall, "Unusual Abnormalities of the Arteries at the Base of the Brain," Journal of Anatomy and Phys., London vol. xiii., p. 396, 1886. The vertebral arteries of either side were joined as usual to constitute the basilar, which immediately after its formation divided into two trunks, and again uniting formed a loop. The loop, which was about two lines in length, was situated near the lower border of the pons Varolii. The main artery now ran forward, giving off the inferior cerebellar and transverse arteries of the pons, also the two superior cerebellar arteries, and then terminated in two small branches in the position of the posterior communicating arteries.

Each internal carotid, after giving off the ophthalmic branch, varied in its arrangement on the two sides. The left carotid gave off first the posterior cerebral artery. This was joined by the small communicating branch from the basilar, and then ran on to its normal distribution in the posterior lobe of the cerebrum. The next branch was the middle cerebral, which was quite normal both in size and course.

The terminal portion of the left internal carotid having the direction and appearance of a large anterior cerebral artery, ran forward to the anterior extremity of the locus perforatus anticus, where it divided into two equal branches. One of these, in direction and appearance the terminal of this vessel, continued forwards in the usual course of the left anterior cerebral artery. The other division took a sharp turn to the right, and then, after a short transverse course of about two and one-half lines, another sharp turn forwards, so as to almost form two right angles where it constituted the anterior cerebral artery of the right side.

The right internal carotid artery resembled the left in giving off both posterior and middle cerebrals. These two arteries had the usual size and distribution. The former received the communicating branch from the right basilar. As the fundamental right anterior cerebral was derived from the left internal carotid, the branch of the right carotid which represented that artery was very slender and joined the functional vessel where it passed forwards into the great longitudinal fissure.

Incoronato, Di un anomalia del poligono arterioso cerebrale. Recherche fatte nel laboratorio de Anatomia normale della R. Università di Roma, nell' anno 1872, p. 95, describes an anomaly³ of the Circle of Willis, in which the carotids unite *directly* with one another instead of doing so by means of the anterior cerebrals and anterior communicating. The posterior communicating are wanting.

³ In this case aneurismal disease at the seat of anastomosis would have inevitably interfered with the chiasm, and in all probability with eye symptoms resembling those in Dr. Mitchell's case.—DERCUM.

Incoronato Anomalia del polygono arteriosa cerebrale, Atti Acad. Med. de Roma, 1878, vol. iu., fasc 2, 16-24, describes an anomaly in which there is a double anterior communicating artery, the anterior being much the larger of the two. Again, the right cavernous carotid instead of communicating with the posterior cerebral by means of a posterior communicating artery, anastomoses by means of a large trunk directly with the basilar which it joins at a point corresponding to the normal division of the latter into the two posterior cerebrals.

Weber, S. G., Abnormal Distribution of Circle of Willis, Bost. Med. and Surg. Jour., vol., cvii., 543. 1882. The two anterior cerebral arteries came so near each other that the anterior communicating was only about one-tenth the usual diameter. The right posterior cerebral artery was twice the usual size or larger.

The left posterior communicating artery was nearly twice the usual size and virtually was the origin of the posterior cerebral. The left posterior cerebral was about half the usual size until it met the posterior communicating. Thus the region supplied by the right cerebral artery received nearly all its blood through the basilar; that supplied by the left posterior artery received more than three-fourths its blood through the left carotid.

The right posterior cerebral was so small that it could not be distinguished from other small and terminal arteries. Its place was taken by one of the branches from the basilar which was larger than usual, and after passing backwards and upwards, occupied the normal position on the posterior cerebellar between the cerebellum and medulla. The basilar artery was larger than usual, the vertebral uniting at a lower level than normal.

The middle cerebral arteries and the anterior cerebral arteries were normal.